LETTERS TO THE EDITOR

● The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.

● All letters must be typed with double spacing and signed by all authors.

● No letter should be more than 600 words.

● In general, no letter should contain more than six references (also typed with double spacing).

10 year review of cardiac tumours in childhood

SIR,—In their review Abushaban and colleagues (*Br Heart J* 1993;70:166-9) found six cases of cardiac tumours in childhood. They conclude that such tumours are extremely rare and that they have an excellent outcome. We identified 15 children with rhabdomyomas who were symptomatic in infancy: 13 had presented by the age of one month.¹ Our results suggest that presentation in infancy, rather than being "uncommon", is probably the rule. Rhabdomyomas usually regress after birth so they are indeed more likely to present in infancy than later.

Two thirds of our patients were male; Harding *et al* in a review of 355 reported cases reported a similar sex distribution.²

Abushaban *et al* may have been fortunate in their patients' outcome: five (33%) of the children we identified had died within one month of birth, three within 3 days. Only one of their four patients with rhabdomyomas had tuberous sclerosis at follow up: whereas 12/15 (80%) of our patients did. It can be difficult to diagnose tuberous sclerosis in the early months of life (when the cardiac lesions are most likely to present). All children with cardiac rhabdomyomas should have careful investigation with prolonged follow up. The report of the isolation of one of the genes for tuberous sclerosis means that molecular analysis may in future allow diagnostic proof of tuberous sclerosis.³

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- 1 Webb DW, Thomas RD, Osborne JP. Cardiac rhabdomyomas and their association with tuberous sclerosis. *Arch Dis Child* 1993;68:367-70.
- 2 Harding CO, Pagon RA. Incidence of tuberous sclerosis in patients with cardiac rhabdomyomas. *Am J Med Genet* 1990;37:443-6.
- 3 The European Chromosome 16 Tuberous Sclerosis Consortium. Identification and characterisation of the tuberous sclerosis gene on chromosome 16. *Cell* 1993;75:1305-15.

Radiation hazards to the cardiologist

SIR,—Professor Camm and his committee have provided an excellent review of the radiation hazards to cardiologists (*Br Heart J* 1993;70:489-96). It covers comprehensively the hazards involved in interventional procedures: but cardiologists may also be exposed to radiation when they perform nuclear cardiology procedures. The hazard to the operator in these procedures is very small compared with that during invasive procedures; none the less, training in nuclear cardiology is an essential part of cardiology training.¹ Experienced cardiologists and those in training must know how to handle unsealed radioactive sources in order to minimise hazard to themselves and to others. Though most cardiologists do not perform both invasive and nuclear procedures themselves, some do.

Consideration of the radiation hazards during nuclear cardiology procedures would therefore have added to the completeness of the review. The British Nuclear Cardiology Group would be happy to contribute to any future review.

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- 1 Joint Training and Manpower Group representing the Specialty Advisory Committee and the Cardiology Committee of the Royal College of Physicians of London, and the British Cardiac Society. Proposals for a new training programme for cardiology. *Br Heart J* 1990;63:317-20.

Pathogenesis of oedema in chronic severe anaemia: studies of body water and sodium, renal function, haemodynamic variables, and plasma hormones

SIR,—I was interested to read the paper by Anand and his colleagues on the pathogenesis of oedema in chronic severe anaemia (*Br Heart J* 1993;70:357-62) that very nicely shows the mechanism for the development of fluid retention and congestion in patients with increased cardiac output. The low peripheral resistance associated with anaemia reduces the blood pressure. This directly reduces salt and water excretion and this reduction will be exacerbated by reflex stimulation of the sympathetic nervous system and renin-angiotensin-aldosterone system as the blood pressure is perceived to be "threatened". I believe a similar mechanism operates in the postpartum heart failure syndrome of Northern Nigeria. In 1979 I and others put forward an almost identical explanation to explain how women with normal or high cardiac outputs, normal left ventricular function, and apparently normal renal function could develop fluid retention and severe circulatory congestion.¹ Like Anand *et al* we suggested that low peripheral resistance and blood pressure (induced in Nigeria by excessive heating due to lying on "hot" beds for over 40 days) caused the kidneys to

retain salt and water even in the face of a high salt intake. Furthermore, in a *Lancet* hypothesis article² I suggested that this was a mechanism that might explain the increased tendency for oedema formation in hotter climates. The same mechanism may be operating in patients with postpartum heart failure elsewhere. More recently a paper from Brazil reported that patients with peripartum cardiomyopathy had normal or high cardiac index, nearly normal left ventricular ejection fractions, and lower systemic vascular resistance than patients with dilated cardiomyopathy.³

All these examples therefore confirm the central hypothesis that it is a low or "threatened" arterial pressure that is the main stimulus for salt and water retention in heart failure⁴ not only in high-output failure caused by excessive vasodilatation but also in low-output failure caused by poor myocardial function.

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- 1 Sanderson JE, Adesanya CO, Anjorin FI, Parry EHO. Postpartum cardiac failure—heart failure due to volume overload? *Am Heart J* 1979;97:613-21.
- 2 Sanderson JE. Oedema and heart failure in the tropics. *Lancet* 1977;ii:1159-61.
- 3 Marin-Neto JA, Maciel BC, Urbanetz LL, Gallo L, Almeida-Filho OC. High-output failure in patients with peripartum cardiomyopathy: comparative study with dilated cardiomyopathy. *Am Heart J* 1991;121:134-40.
- 4 Harris P. Role of arterial pressure in the oedema of heart disease. *Lancet* 1988;i:1036-8.

BRITISH CARDIAC SOCIETY NEWSLETTER

While the difficulties of implementing the Calman proposals continue to occupy a considerable amount of time, another initiative is beginning to surface—namely, continuing medical education (CME). The joint declaration from the Royal Medical Colleges and their faculties makes a clear commitment to the introduction of CME for all doctors. The British Cardiac Society is cooperating with the Royal Colleges in discussing the quantity and quality of CME. It is likely to require a commitment from individual doctors to participate both in internal CME at their normal place of work and a requirement to attend a certain amount of CME externally. The programmes for CME should be developing over the next year or so and the British Cardiac Society is keen to participate in the process.

Your President was invited to the 60th Anniversary Meeting of the Netherlands Cardiac Society in April in Amsterdam, and contributed to the programme reviewing the development of cardiac surgery in Europe.